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Case report

Unreported cardiac arrhythmias in aluminium worker

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ABSTRACT

Aluminium (Al) is the third most prevalent element, representing approximately 8% of total mineral components in the earth's crust (1). Chronic exposure to Al is mainly encountered at particular work places, for example, in foundries or in the Al powder industry, as an occupational exposure. In case of occupational Al exposure, inhalation is the main route of uptake. Chronic exposure to Al is associated with skeletal, neurological, hematological and lung changes. Studies regarding the Al powder industry showed that long-term inhalative exposure to Al can induce pulmonary fibrosis (2). Although there is only one report about ventricular tachycardia as a cardiac manifestation in occupationally exposed persons (3), in this report, we presented a case that had Mobitz type I second-degree atrioventricular block and nonsustained ventricular tachycardia. To our knowledge, this is the first report in chronic poisoning.

1. Introduction

Aluminium (Al) is the third most prevalent element, representing approximately 8% of total mineral components in the earth's crust.[1] Chronic exposure to Al is mainly encountered at particular work places, for example, in foundries or in the Al powder industry, as an occupational exposure. In case of occupational Al exposure, inhalation is the main route of uptake. Chronic exposure to Al is associated with skeletal, neurological, hematological and lung changes. Studies regarding the Al powder industry showed that long-term inhalative exposure to Al can induce pulmonary fibrosis.[2] Although there is only one report about ventricular tachycardia as a cardiac manifestation in occupationally exposed persons,[3] in this report, we presented a case that had Mobitz type I second-degree atrioventricular block and nonsustained ventricular tachycardia. To our knowledge, this is the first report in chronic poisoning.

2. Case report

A 42-year-old male patient – who formerly did not have any known disease – was admitted to emergency department of our hospital due to dizziness. His medical history showed that he had been exposed to aluminium steam in foundry work for 10 years. On his physical examination, blood pressure was 110/70 mmHg and pulse rate was arrhythmic with 60 beats/min. Electrocardiography showed that he had Mobitz type I second-degree atrioventricular block (Fig. 1) and nonsustained ventricular tachycardia (Fig. 2). Blood tests were found normal except microcytic anemia. Serial creatinine kinase and troponin I were within normal limits. We did not find any electrolyte imbalance. A transthoracic echocardiogram was normal with left ventricular ejection fraction 60% and no significant valvar regurgitation. We performed coronary angiography to exclude ischemia and the results were normal. Supraventricular and ventricular tachycardia were not induced during electrophysiological investigation. Since he had no symptom and he was normotensive, we observed the patient without pacing.

Blood aluminium level was found 23.6 ug/L (reference range 0–14 ug/L) by atomic absorption method. We excluded lead intoxication which is also encountered in case of occupational Al exposure.

We considered this patient as an occupationally exposed person by reason of breathing aluminium steam for 10 years and having anemia.

Electrocardiographic findings were improved in the seventh day of hospitalization by the administration of 600 mg/day N-acetylcysteine (NAC) and hydration. 24-h Holter electrocardiogram recording revealed that he had sinus rhythm (average heart rate 74) with only four Mobitz type I second-degree atrioventricular block. He is asymptomatic and we advised him to change his work.

3. Discussion

Aluminum is increasingly used for the manufacture of many kinds of vehicles and in the construction industry due to its light weight, high durability and high electrical and thermal

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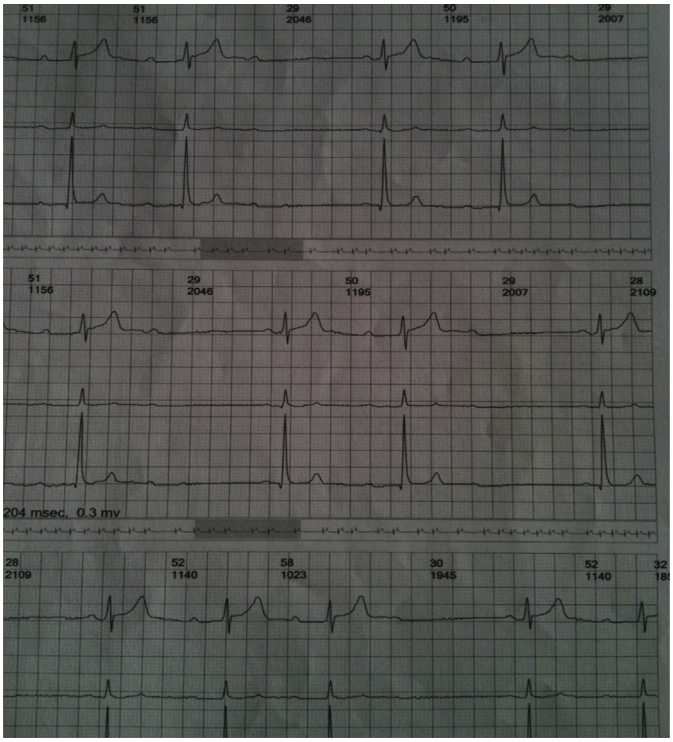

Fig. 1. Electrocardiography revealed Mobitz type I second-degree atrioventricular block.

conductivity. In case of occupational Al exposure, inhalation is the main route of uptake. Therefore, toxicity is dependent on the species and particle size of the inhaled material.[1]

Aluminium phosphide poisoning has a high mortality risk due to cardiovascular involvement. There are some reports about serious toxic effects of acute Al poisoning. Siwach and et al.[4] reported 30 patients of acute Al poisoning. They observed life threatening ventricular tachycardia in 12 patients and ventricular fibrillation in 7 patients. One-third of the patients developed variable degrees of heart block. They accused toxic myocarditis that resulted from phosphine of these arrhythmias. In presented case, who have exposed Al for 10 years, we found serial cardiac markers such as troponin, creatine kinase-MB and echocardiographic investigation normal.

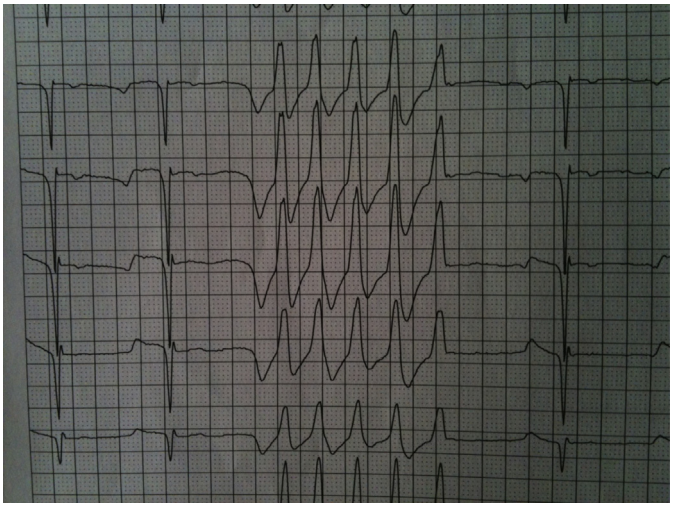

Fig. 2. Electrocardiography showed nonsustained ventricular tachycardia.

Soltaninejad and et al.[5] evaluated the frequency of cardiac manifestations and electrocardiographic findings during acute AlP poisoning. They observed dysrhythmia in nine cases. Of them, seven patients showed atrial fibrillation and two cases had junctional rhythm. Additionally, they found that there was a significant correlation between cardiac dysrhythmia and mortality.

In our patient, we found microcytic anemia in blood tests. The current concept about the pathogenesis of aluminum-related microcytosis is that Al might interfere with iron kinetics at different levels, including alterations in delta amino levulinic dehydrase activity and intermediate porphyrin products and impaired intestinal iron absorption and cellular uptake.[6,7] In conclusion, Al inhibits erythropoiesis and iron metabolism, probably hinders hemoglobin synthesis and erythroid cell maturation. Besides chelation therapy with deferoxamine, erythropoietin may prevent the oxidative stress due to Al exposure.[8] Chronic Al exposure may lead to biochemical and morphological alterations similar to premature erythrocyte death (eryptosis), induced by oxidant compounds in human erythrocytes. Irrespective of the antioxidant mechanism, erythropoietin treatment benefits patients with anemia associated to altered redox environment.

An experimental study which was conducted by Lall and et al.[9] showed that the effect of Al on heart is mediated by both declined cellular metabolism of the myocardium and by necrosis of the cardiac tissue resulting in the release of reactive oxygen intermediates. Tehrani and et al.[10] showed the benefit of NAC in patients with acute aluminum phosphide poisoning. Additionally, NAC was found to be beneficial in rats. Treatment with NAC improved the survival due to the reduction of myocardial oxidative injury in this experimental study.[11,12] The administration of a loading dose of NAC could treat the arrhythmia. Although these findings are valid in acute poisoning, we administered 600 mg/day NAC with hydration. On the seventh day of therapy, we performed 24-h Holter electrocardiogram recording. It revealed that he had sinus rhythm (average heart rate 74) with only four Mobitz type I second-degree atrioventricular block.

Other agents include trimetazidine, which switches myocyte metabolism to glucose from fatty acids, thus reducing oxygen consumption, and may have a potential role.[13]

In conclusion, Mobitz type I second-degree atrioventricular block might develop in occupationally exposed persons as well as nonsustained ventricular tachycardia. Additionally, since the correlation between cardiac dysrhythmia and mortality observed in previous studies, a clinical trial on the effectiveness of prophylactic administration of NAC in acute aluminum intoxication should be considered.

Ethical approval

None.

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None.

Author contributions

All authors contributed to research design, or the acquisition, analysis or interpretation of data, to draft the paper and approved the final version.

Conflict of interest

None declared.

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